example, rates of assault on public transport in three Dutch cities were reduced when 1200 unemployed young people were hired as inspectors.[17] Similarly, the installation of closed circuit television on the London underground has reduced muggings and theft. Neighbourhood watch schemes have, however, resulted in few measurable falls in crime.

Interestingly, it is in situational crime prevention that the adoption of violence as a public health issue is having greatest effect. In the United States case-control studies have shown an increased risk of homicide and suicide in homes where firearms are available. The availability of handguns was responsible for a sevenfold difference in the rate of homicide between Seattle, in the United States, and Vancouver, in Canada, despite a similar incidence of assault in the two cities[18]—a finding that was influential in the success of the Brady bill on gun control in the United States. Public health approaches have also helped to define risk of injury due to assault in relation to alcohol consumption. In urban violence in Britain consumption of more than 10 units of alcohol in a six hour period has been linked to more severe injury, and consumption of 8 to 15 units has distinguished injured from uninjured men in the same environments.[19]

As the consequences of violence become more apparent in terms of increased morbidity and cost the need for doctors to join forces with social scientists to tackle this problem becomes ever more obvious. Evidence also exists that, independently of socioeconomic variables, injury in violent crime is linked to adolescents with a history of drug misuse, elective surgery, and trauma.[20] Preventing crime and violence should be a central issue in health care.

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1 Yoshikawa H. Prevention as cumulative protection: effects of early family support and education on chronic delinquency and its risks. *Psychol Bull* 1994;115:28-54.
2 Home Office. *Criminal statistics, England and Wales, 1991*. London: HMSO, 1993.
3 Shepherd JP, Ali MA, Hughes AO, Levers BGH. Trends in urban violence: a comparison of accident department and police records. *J R Soc Med* 1993;86:87-9.
4 Mercy JA, O'Carroll PW. New directions in violence prediction: the public health arena. *Violence and Victims* 1988;3:285-301.
5 Shepherd JP, Farrington DP. Assault as a public health problem. *J R Soc Med* 1993;86:89-92.
6 Farrington DP, West DJ. The Cambridge study in delinquent development: a long-term follow-up of 411 London males. In: Kerner HJ, Kaiser G, eds. *Criminality: personality, behaviour, life history*. Berlin: Springer Verlag, 1990:115-38.
7 Lauritsen JL, Laub JH, Sampson RJ. Correctional and delinquent activities: implications for the prevention of violent victimisation among adolescents. *Violence and Victims* 1992;7:91-108.
8 Farrington DP, Loeber R, Van Kammen WB. Long-term criminal outcomes of hyperactivity-impulsivity, attention deficit and conduct problems in childhood. In: Robins LN, Rutter M, eds. *Straight and devious pathways from childhood to adulthood*. Cambridge: Cambridge University Press, 1990:62-81.
9 Masten AS. Resilience in development: implications of the study of successful adaptation for developmental psychopathology. In: Cicchetti D, ed. *Rochester symposium on developmental psychopathology*. Vol 1. *The emergence of a discipline*. Hillsdale, NJ: Erlbaum, 1989:261-94.
10 Farrington DP. Early developmental prevention of juvenile delinquency. *Criminal Behaviour and Mental Health* 1994;4:204-27.
11 Clarke RV. Situational crime prevention: achievements and challenges. In: Tonry M, Farrington DP, eds. *Building a safer society*. Chicago: University of Chicago Press (in press).
12 Berrueta-Clement JR, Schweinhart LJ, Barnett WS, Epstein AS, Weikart DP. *Changed lives: the effects of the Perry preschool program on youths through age 19*. Ypsilanti, MI: High/Scope Press, 1984.
13 Johnson DL, Walker T. Primary prevention of behavior problems in Mexican-American children. *Am J Community Psychol* 1987;15:375-85.
14 Lally JR, Mangione PL, Honig AS, Wittner DS. More pride, less delinquency: findings from the ten-year follow-up study of the Syracuse University family development research program. *Zero to Three* 1988;April:13-8.
15 Olds DL, Henderson CR, Chamberlain R, Tatelbaum R. Preventing child abuse and neglect: a randomised trial of nurse home visitation. *Pediatrics* 1986;78:65-78.
16 Poyner B, Webb B. *Successful crime prevention: case studies*. London: Tavistock Institute of Human Relations, 1987.
17 Van Andel H. Crime prevention that works: the case of public transport in the Netherlands. *British Journal of Criminology* 1989;29:47-56.
18 Sloan JH, Kellerman AL, Reay DT, Ferris JA, Koepsell T, Rivara F, *et al*. Handgun regulations, crime, assaults and homicide. *N Engl J Med* 1988;319:1256-62.
19 Shepherd JP. Violent crime: the role of alcohol and new approaches to the prevention of injury. *Alcohol Alcohol* 1994;29:5-10.
20 Shepherd JP, Peak JD, Haria S, Sleeman D. Characteristic illness behaviour in assault victims: DATES syndrome. *J R Soc Med* 1995;88:85-7.

Biological influences on criminal behaviour: how good is the evidence?

Available studies have their limitations

The perception that crime, especially violent crime, has become one of the most serious problems facing society has led to determined efforts by many researchers to find the causes of criminal behaviour. Researchers have focused on biological causes, believing that a biological basis of criminality exists and that an understanding of the biology will be useful in predicting which people are predisposed to become criminals. In the 1960s it was proposed that males with an extra Y chromosome were predisposed to violent criminal behaviour; later work found no support for this hypothesis.[1] Recently, two approaches, one genetic, the other biochemical, have received widespread publicity. I would argue that currently neither approach provides convincing evidence that criminal behaviour can be understood in terms of genetics or biochemistry.

Before these two approaches are discussed, the many family, twin, and adoption studies that have concluded that a biological basis exists for antisocial behaviour should be noted.[2-4] At least two recent reviews, however, have suggested that the support for these conclusions, especially those concerned with violent crime, is not strong. A meta-analysis of the literature found only a "low-moderate correlation" between heredity and crime.[5] Moreover, the "better designed and more recently published studies provided less support for the gene-crime hypothesis than more poorly designed and earlier published investigations."[5] And a review published last year concluded: "Together, the data do not suggest a strong role for heredity in violence."[6]

For about 25 years researchers have reported correlations between low cerebrospinal fluid concentration of 5-hydroxyindoleacetic acid, a metabolite of the neurotransmitter serotonin, and violent and criminal behaviour. Although more than 100 studies have been published on this topic, later studies cannot be regarded as confirming the results of earlier ones. The behaviour characteristically associated with low concentrations of the metabolite has shifted from depression to general aggressive behaviour to impulsive aggressive behaviour.[7] The later studies, which have used more refined definitions than earlier ones, therefore do not replicate the earlier ones.[8]

Even if an association was established between low 5-hydroxyindoleacetic acid concentration in cerebrospinal

fluid and some well defined violent or criminal behaviour, causation is not proved. Do low concentrations of the metabolite cause the abnormal behaviour or does the abnormal behaviour trigger physiological responses in the body that lower the concentrations? Perhaps there is some third factor —biological, psychological, or environmental—that is the underlying cause of both the low concentrations and abnormal behaviour.

Within the past year Brunner and colleagues have reported an association between a point mutation in the structural gene for monoamine oxidase A in a large Dutch family and aggressive criminal behaviour among many males in that family.[9] Other single gene conditions are known to result in abnormal behaviour—for example, the symptoms of Wilson disease, which result from accumulation of copper primarily in the liver and brain, can mimic those of schizophrenia. The gene for monoamine oxidase A, however, is the first instance of an altered gene being implicated in specifically criminal aggressive behaviour. As monoamine oxidase A is involved in serotonin metabolism researchers have suggested that the gene mutation that results in monoamine oxidase A deficiency is related to the low cerebrospinal fluid concentrations of 5-hydroxyindoleacetic acid found in association with criminal behaviour.[9]

Although the defect in the gene for monoamine oxidase A is likely to be responsible for the learning disabilities and possibly the abnormal behaviours in the Dutch family, there is little prospect that a better understanding of this condition will improve our understanding of criminality. The primary effect of the mutation is learning disability; the aggressive behaviour, which does not appear in all the males with the genetic abnormality, may result from the learning disability and its attendant problems rather than directly from the altered gene. Furthermore, as the authors point out, this genetic defect is extremely rare. Even if its importance in causing criminal behaviour is confirmed it is unlikely to be important in more than a minute proportion of criminals.

The genetic and biochemical studies discussed above found an association between a biological factor and violent criminal behaviour. But a series of adoption studies in which the criminal history of an adopted male was compared with the criminal history of both his biological and his adoptive fathers found that genetic influences were significant in cases of property crime but not in cases of violent crime.[10] This conclusion is as problematic as finding a genetic basis for violent crime. Adoption studies have their own methodological problems—for example, various social characteristics of the adoptive and biological home environments are correlated, and adoptive parents are generally of higher socioeconomic status than parents in general.[11] Although researchers try to correct for these complicating factors, the corrections are post hoc.

Although studies of the biological basis of violence interest nearly everyone, currently, research on this topic is far too preliminary to be of use to psychiatrists treating people who are predisposed to or engage in violent and criminal activities. Nevertheless, there have been suggestions that public policy should be informed by the results of these biological studies. Given the extremely tentative nature of their conclusions and the far reaching consequences of labelling people as potential criminals on the basis of some biological marker, an awareness of the limitations of these studies is crucially important.

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1 Borgaonkar DS, Shah SA. The XYY chromosome male—or syndrome. *Prog Med Genet* 1974;10:135-222.
2 Christiansen KO. A review of studies of criminality among twins. In: Mednick SA, Christiansen KO, eds. *Biosocial bases of criminal behavior*. New York: Gardner Press, 1977:45-88.
3 Cloninger CR, Gottesman II. Genetic and environmental factors in antisocial behavior disorders. In: Mednick SA, Moffitt TE, Stack SA, eds. *The causes of crime: new biological approaches*. New York: Cambridge, 1987:92-109.
4 Rowe DC. Genetic and environmental components of antisocial behavior: a study of 265 twin pairs. *Criminology* 1986;24:513-32.
5 Walters GD. A meta-analysis of the gene-crime relationship. *Criminology* 1992;30:595-613.
6 Carey G. Genetics and violence. In: Reiss AJ Jr, Miczek KA, Roth JA, eds. *Understanding and preventing violence*. Vol 2. *Biobehavioral influences*. Washington, DC: National Academy Press, 1994:21-58.
7 Van Praag HM. Serotonergic dysfunction and aggression control. *Psychol Med* 1991;21:15-9.
8 Kidd KK. Association of disease with genetic markers: deja vu all over again. *Am J Med Genet (Neuropsychiatric Genetics)* 1993;48:71-3.
9 Brunner HG, Nelen M, Breakefield XO, Ropers HH, van Oost BA. Abnormal behavior associated with a point mutation in the structural gene for monoamine oxidase A. *Science* 1993;262:578-80.
10 Brennan PA, Mednick SA. Genetic perspectives on crime. *Acta Psychiatr Scand* 1993;370(suppl): 19-26.
11 Billings PR, Beckwith J, Alper JS. The genetic analysis of human behavior: a new era? *Soc Sci Med* 1992;35:227-38.

Television violence and children

Its effects need to be seen in the context of other influences on children's mental health

Children watch two to three hours' television daily from the age of 3 or earlier, and during childhood they average more time in front of the television set than in the classroom.[1] Although watching television has positive effects,[2] attention is usually focused on its negative ones. Some people believe that children may be harmed by watching violence on the screen,[3,4] and, although over 1000 research studies have established an association between screen violence and the level of aggressive behaviour in some children and young people,[5,6] causation has not been established. Increased aggression may not be the only negative effect. Children may find some of the images frightening and in rare cases develop anxiety and phobic reactions; more commonly they may develop short lived nightmares and other sleep problems.[7]

Television violence may influence children in four ways: making them want to imitate what they see, reducing learnt inhibitions against violent behaviour, desensitising them to violence through repetition, and increasing arousal.[1] Viewing violence on the screen does not on its own cause violent behaviour.[8] The most plausible model to explain the association between viewing violence and increased aggression is an interactive one in which viewing and aggression affect each other and, in turn, are stimulated by other related variables.[9]

Aggression as a problem solving behaviour is learnt early in life, is usually learnt well, and is resistant to change.[10] Individual variation in the level of aggressive behaviour and violence in children, adolescents, and adults depends on many interacting factors, of which media influences are likely to be less important than constitutional, parental, educational, and other environmental influences. Contributing factors include being the victims of violence and bullying and witnessing violence perpetrated against others, especially at home. The emphasis on establishing whether television violence and actual violence are related has resulted in the neglect of these other, more important influences on the development of aggressive behaviour and other effects of